TABLE V.—FREQUENCY OF EMPYEMA IN PNEUMONIA.

		Per cent.
Number of empyemata following bronchopneumonia	5	1.8
Number of empyemata following croupous pneumonia	22	7.5
Number of empyemata following unspecified pneumonia	12	
Total number of empyemata in 611 cases of pneumonia	39	

TABLE VI.—CORRECTED PERCENTAGE OF INCIDENCE OF EMPYEMA.

Percentage of empyemata after bronchopneumonia (deaths during acute pneumonia process substracted)	3.14
Same correction for croupous pneumonia	8.59

TABLE VII.—MORTALITY IN POSTPNEUMONIC EMPYEMA.

		Per cent.
Number of deaths following bronchopneumonia empyemata	4	80.0
Number of deaths following croupous pneumonia empyemata	8	36.3
Total number of deaths in 39 cases of empyema	15	38.4

A slight discrepancy in a comparison of the figures of the various tables is due to the omission of cases in which all the data necessary was not present in the history.

UNKNOWN FORMS OF ARTERITIS, WITH SPECIAL REFERENCE TO THEIR RELATION TO SYPHILITIC ARTERITIS AND PERIARTERITIS NODOSA.

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Comparatively little is known about arteritis and its significance if we disregard syphilitic endarteritis, which is well known, and tuberculous arteritis in the meninges associated with exudation, which is the dominant feature. Otherwise but little has been written about arteritis until rather recently. According to Spiro,[1] French authors, and later Wiesel and v. Wiesner, described peculiar inflammations or rather necroses in the small arteries, particularly in the heart, in measles, scarlet fever, diphtheria, suppurative processes, etc., which result in scars and are believed to favor the development of coronary sclerosis, but apparently these observations have attracted but little attention. More recently the vascular changes in infections and intoxications have been studied more closely. Thus Herzog[2] describes the following changes in poisoning with illuminating gas: Hyaline swelling of the walls of the small arteries in the brain with proliferation of the endothelium and hyaline thrombosis, followed later by calcification and softening of the brain tissue. Similar changes with calcification have been described as occurring quite early by Geipel and Schmorl,[3] the small arteries in the lenticular nucleus being especially involved. Similar changes may occur in other forms of poisoning, *e. g.*, hydrocyanic acid, salvarsan and phosgen gas, but it really does not concern any arteritis.

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[1] Virchow's Arch., 1920, No. 227.

[2] München. med. Wchnschr., 1920, No. 19.

In various infectious diseases inflammatory vascular changes may occur, as seen especially in the cerebral vessels in influenza with hemorrhages, which, however, seem to depend largely on degenerative changes.[4] Markus[5] describes infiltration with cells in the wall of the vessels in the pia in influenza, and Stoerck and Epstein[6] describe necrosis and degeneration with reparatory processes in influenza, resembling the changes in arteriosclerosis. Of greater interest are the changes that have been found in the vessels of the skin in typhus by Frankel[7] and others. Nicol[8] describes these changes as involving the capillaries and precapillary arteries in most of the organs, with partly exudate, partly proliferative changes as well as necrosis. Inflammatory and degenerative changes occur also in the small vessels in the skin in epidemic meningitis, and Weiss and Stufeland[9] describe certain changes in the capillaries of the skin in scarlet fever.

While engaged in the examination of microscopic preparations from a large amount of anatomical material I met with occasional peculiar lesions in the vessels that do not correspond to the usual forms, and on this account I have thought it worth while to describe some of the cases with some detail.

CASE I.—*Idiopathic Renal Hemorrhage.* This concerns a kidney which was removed from a man, aged twenty-two years, who for about one year had suffered from frequent hemorrhages from the one kidney and later from pain. Macroscopically the kidney appeared normal, but microscopically there were found necrotic foci throughout the kidney, in the center of which were arteries with swollen walls and narrowing of the lumen. The swelling was due to proliferation of the intima and to hyaline changes in the connective and elastic tissue, while the surrounding renal tissue frequently was necrotic, there being, however, no sign of inflammation.

The cause and nature of the changes in the arteries were obscure. Syphilis was excluded and it hardly concerned arteriosclerosis.

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[3] München. med. Wchnschr., 1920, No. 43.
[4] Harbitz: Norsk. Mag. f. Lagevidenskapen, 1920.
[5] Ztschr. f. Neurol. u. Psych., 1920.
[6] Frankf. Ztschr. f. Pathol., 1920, No. 23.
[7] München. med. Wchnschr., 1914, 1915, 1917.
[8] Ziegler's Beiträge, 1919, No. 65.
[9] Ibid., 1918.

The case might be regarded as a local form of periarteritis nodosa, especially in view of the necroses in the media and the proliferation of the intima. That the lesion of the arteries, which involved also vessels in the papillæ, was the cause of the hemorrhage seems altogether reasonable, and in that case it hardly would be correct to speak of the case as one of "idiopathic hemorrhage," as the hemorrhages had an organic basis.

CASE II.—*Purpura Rheumatica.* This concerns a man, aged forty years, who died with the clinical picture of "purpura rheumatica" about two months after the beginning of the illness. At postmortem there was found extensive thrombosis (inferior vena cava, in the left iliac and the left femoral vein, hepatic vein with infarct in the liver, and also in several sinuses of the dura with large hemorrhagic infarcts in the brain). In the skin there was no evident exanthem, but extensive infiltrations of leukocytes, lymphocytes and red corpuscles as well as pure hemorrhages. There was also extensive changes, in the smaller bloodvessels and arteries as well as veins, with cellular infiltrations in the walls and necrosis. The intima was swollen and there was often thrombosis. These changes were most marked in the arteries, medium sized and small. The veins were not so markedly involved, but there was more or less necrosis and infiltration as well as thrombosis.

In this case syphilis may be excluded. The cutaneous infiltrations and the thrombosis were more extensive than is the case in exanthematic typhus, a disease that so far as known has not occurred in Christiania for many years. As regards periarteritis nodosa it should be noted that there were no typical nodular formations in the walls of the vessels and that the infiltrations were larger and more extensive than is the case in that disease. Hence it seems most reasonable to assume the presence of some unknown form of infection. Bacteria were not demonstrable by the usual methods.

CASE III.—*Peculiar Disease of Cerebral Arteries* (Figs. 1 to 3) A woman, aged twenty-six years, of doubtful habits. She had suffered much with headache since early in life; during the last two years she had had attacks of fainting off and on, and about a year and a half ago she received a blow to the head during such an attack. In the autumn of 1917 the hands became tremulous; even earlier she had had double vision and sometimes vestibular attacks, with vomiting as well as convulsive movement of the face. In the autumn of 1918 she had influenza and became worse afterward, with pain in the legs, difficulty in walking and convulsive attacks of a hysterical nature. In March, 1919, she was examined by Dr. Svenson, who reports as follows: Dull; rather stuporous; somewhat disoriented; speech monotonous; eyes normal; no symptoms on the part of the cranial nerves, pons or bulb. Reduced strength in the arms; ataxia; spastic paraparesis of both legs with increased patellar reflexes and positive Babinsky sign; Romberg's symptom present; uncertain, spastic walk, but no disturbances of sensibility.

She became bed-ridden, with increasing stupor, headache, choked disk and increasing paresis in the extremities.

The disease was first regarded as a multiple sclerosis, but later, as signs of increasing brain-pressure developed, a tumor of peculiar localization and secondary hydrocephalus was considered. The patient became finally completely comatose, with muscular rigidity, fever, albuminuria and pneumonia. Death on May 19, 1919.

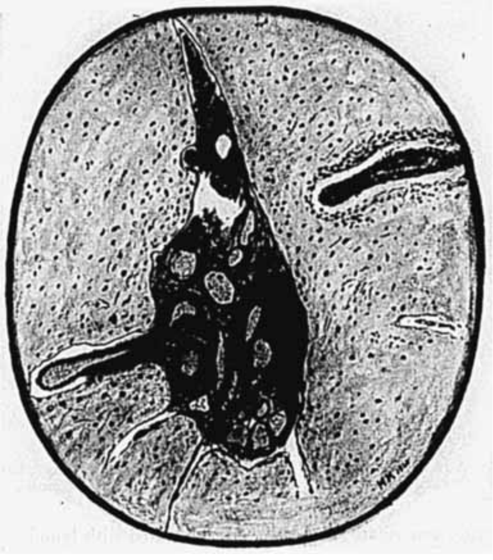

FIG. 1.—Extensive infiltration in the bottom of a sulcus, especially about the arteries, whose walls are thick and partly necrotic; some giant cells.

Only the brain was examined. There was injection of the membranes. In the floor of the lateral ventricles, particularly the left, were areas of a solid, rather tumor-like infiltrations; in the central ganglia were recent areas of hemorrhage, of the size of hemp seeds, and in the centrum semiovale on the right side was a hemorrhage as large as a hen's egg, the surrounding tissue being apparently hardened; there were diffusely infiltrated areas in the corpus callosum also.

The microscopic examination revealed an inflammatory process in the pia arachnoid and in the brain closely related to the bloodvessels, particularly the arteries, and following the bloodvessels from the pia inward into the brain substance. The changes were present seemingly everywhere, also at the base and in marked degree in the central ganglia. There was a remarkable widening of the bloodvessels with hemorrhagic infiltration in various places in the brain. In the pia arachnoid the inflammation was closely related to the smaller arteries and their branches into the cortex, there being no diffuse changes. The inflammatory cells were principally lymphocytic. The process extended inward from the sulci; the veins were dilated and the walls infiltrated with lymphocytes, usually diffusely, but the arterial walls were more involved, being thickened, infiltrated with lymphocytes and leukocytes as well as larger cells at the same time as the connective-tissue cells in the intima and of the adventitia had proliferated, the result being a thick ring around a wide lumen. In the dense infiltration were many larger and smaller multinucleated giant cells. There was no marked necrosis. In many places the process extended throughout the whole segment of the vessel, but in other places the segment was only partially involved. Smaller arteries might be practically closed and in the remnant of the lumen thrombi were present. Among the epithelioid cells were lymphocytes and also different

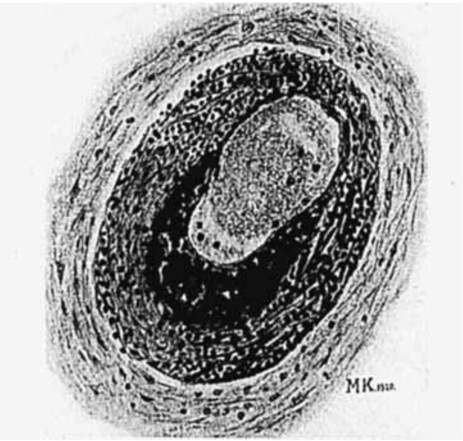

FIG. 2.—A smaller arterial branch in the surface of the brain severely infiltrated, structure not clear, partly obliterated, necrosis of intima and media.

kinds of plasma cells as well as yellow iron-containing pigments in amorphous masses or enclosed in larger cells. In the central ganglia there was the same picture with in places pure hemorrhages. The lumen of the arteries might be filled with epithelioid and giant cells, the process being perhaps more intense here than elsewhere, usually focal but also more diffuse throughout the arterial walls as well as beyond. The cells were mostly mononuclear, but there were also plasma cells and polymorphonuclear leukocytes, sometimes in large masses. There were no necrotic foci, but in the neighborhood of the severely involved vessels the ganglia cells showed degeneration in varying degrees.

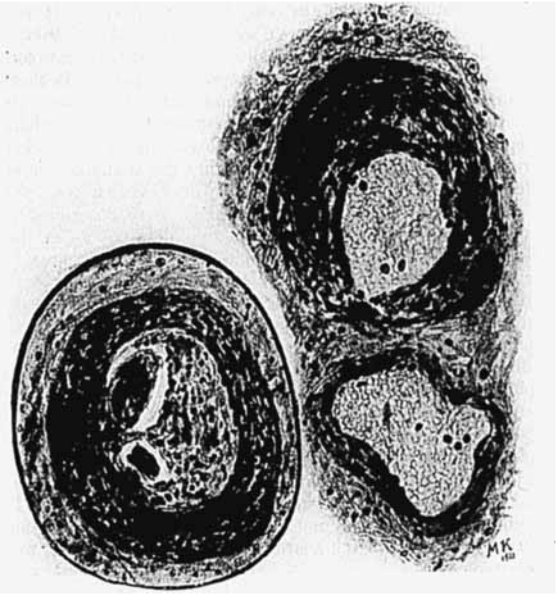

FIG. 3.—The vein (below to the right) shows some inflammation, but the two arteries are extensively infiltrated and there is proliferation, especially in the intima of one of them; in the other the lumen is closed and there are two giant cells among the epithelioid cells; some necrosis in the media.

No microbes could be demonstrated in the sections (Loeffler's method, Gram's method, stains for the tubercle bacillus and Levaditi's method for spirochetes).

In this case we have a chronic cerebral disease in a woman, aged twenty-six years, of uncertain nature, so that no definite clinical diagnosis could be made, although the manifestations clearly pointed more and more to organic brain lesion. To the naked eye the appearances in the brain were also puzzling, their being no definite meningitis, the most striking feature in the brain substance being larger and smaller hemorrhages and infiltrations both on the surface and in the deeper parts. The microscopic examination demonstrated a diffuse and marked inflammatory process connected especially with the arteries, the essential lesion being arteritis, but of what kind? It would seem that the anatomical picture does not correspond to that of any known disease. Lethargic encephalitis is different both as to localization and the kind of inflammatory process, especially in that in this disease it does not concern a primary arterial affection. And then the clinical picture does not correspond to that of lethargic encephalitis. Cerebral syphilis must be considered: Spirochetes were not found, and it is noteworthy that the anatomical lesions differ from those of syphilis. The history does not indicate syphilis (Wassermann's test was negative). There were no gummatous changes. Syphilitic endarteritis, as a rule, does not appear as a diffuse process being generally confined to the larger branches, particularly at the base, and to limited vascular areas, being accompanied generally with thrombosis and narrowing, so that necrosis and softening of the corresponding parts of the brain take place. The hyperemia, the dilatation and infiltration of the veins and the multiple hemorrhages do not correspond to the usual picture of syphilis of the cerebral vessels. The absence of necrosis and caseation speak against syphilis while the marked proliferation of the intima with formation of giant cells remind one somewhat of syphilitic endarteritis. At the same time the intima did not show the marked proliferation usually found in syphilis and, on the other hand, the media was more torn apart than is usual in syphilis. The possibility of syphilis may not be excluded, but the entire clinical picture, together with the anatomical lesions, must be said to go against syphilis.

In periarteritis nodosa the clinical phenomena are variable. Practically all organs may become involved. The duration in this case is longer than seen in periarteritis nodosa, but of course that does not exclude it. It is more important to note that the cerebral arteries are rarely involved in periarteritis nodosa, only two cases being described in the literature. Chovstek and Wechselbaum[10] in 1877 described a case in a soldier, aged twenty-three years, who died after seven months of illness with marked cerebral symptoms and in whom the principal cerebral arteries showed multiple aneurysms. And Müller[10] records a similar observation in a man, aged thirty-two years, who was ill for six or seven months with cerebral and ocular symptoms. To the naked eye there were only scattered hemorrhages of the brain, but microscopically there was inflammatory infiltration in the walls of the vessels with narrowing of the lumen and necrosis and hyaline degeneration of the walls; the arteries in other organs were involved in the usual manner. None of these cases, however, resemble the one I have described when close comparison is made. Furthermore, there were no nodules or aneurysms in my case. The marked thickening and necrosis in the media, which is so characteristic of periarteritis nodosa, being regarded by many as the primary anatomical lesion, was absent in my case.

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[10] Müller, P., Festschr. z. Feier d. 50 jährigen Bestehen der Stadtkrankrah., Dresden-Friedrichsstadt, 1899.

While neither syphilis nor periarteritis nodosa, as a rule, does not show the anatomical lesions described in this case, they may hardly be definitely excluded, especially not syphilitic endarteritis.

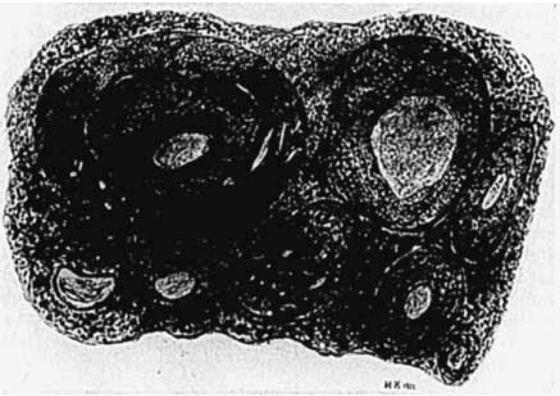

FIG. 4.—Extensive inflammatory changes in small veins and arteries in the bottom of a sulcus, with narrowing and closure of the lumen.

CASE IV.—*Cerebral Arteritis with Mental Disturbances* (Figs. 4 to 6). A sailor, aged forty-six years, was placed in an insane hospital (Dr. Platou) in January, 1919, on account of confusion and hallucinations. He made peculiar movements, *e. g.*, swimming movements, both when standing and when lying down, and would have attacks of violence for a day or so followed by periods of mental clearness, and in the latter part of March he was discharged as cured. It was thought that alcoholism was at the bottom of his trouble.

In June he began to suffer from attacks of fainting, dizziness, vomiting, and later of impulsive violence, when he wanted to kill his sister, and in August he was again confined. He was restless and talked a great deal of nonsense; the right pupil was larger than the left, but reacted to light and accommodation; a little later the left face seemed to be paralyzed. He had had chancre in 1904, but the Wassermann was negative. In the middle of September he

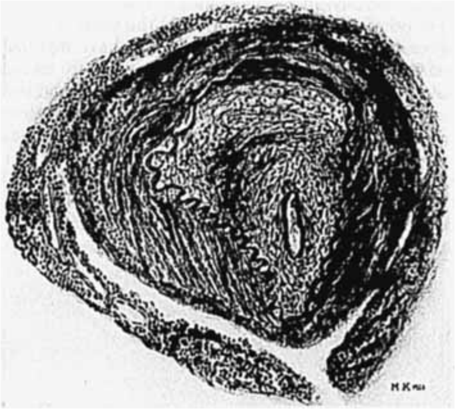

Fig. 5.—All layers of arterial branch extensively involved.

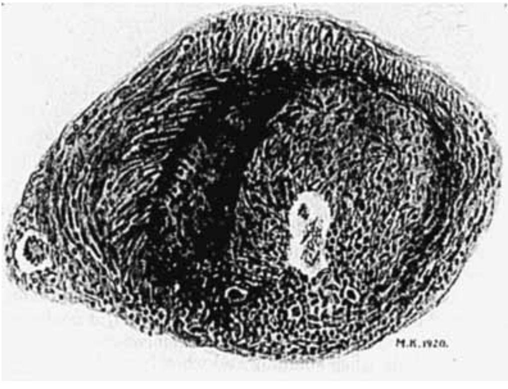

Fig. 6.—Note giant cells and extensive infiltration.

was completely disoriented, could hardly walk and was soporous. Death on September 30.

No definite diagnosis was made, but it was thought that the psychosis depended on an organic brain disease.

The postmortem showed no special changes in the heart or arteries; mucopurulent bronchitis in the right lung; liver and kidneys normal; no special changes in the lower urinary tract or in the digestive organs; no tuberculosis anywhere.

The dura showed a growth as large as a hazelnut extending inward into the brain in gyrus centr. ant. sin. The pia arachnoid was thick and adherent. In the upper part of the left temporal lobe was a soft mass including a nodule as large as a walnut; there were several growths of hazelnut size in both hemispheres, of firm consistency; no sclerosis in the vessels; ventricles not dilated.

Some of the growths were subjected to careful microscopical examination. It was found that vessels extended into the center of the growths; the vessels were thick and the lumen narrowed, in places thrombotic. There were no tubercles or larger caseous or gummatous infiltrations as are characteristic of syphilitic meningo-encephalitis. It concerned an inflammatory process concentrated about and proceeding from the bloodvessels and extending into the brain substance, which was edematous and hyperemic, with dilated vessels, and an inflammatory infiltration extended outward into the brain substance from the affected vessels. It was characteristic that these infiltrations all involved the surface of the brain, or rather the pia arachnoid, from which the process extended into the brain. The process may be described more minutely as follows: From the surface of the brain the infiltration would pass down between two gyri, increase in thickness so that it would form a mass in the bottom of the sulcus and extend into the substance along the vessels; in the center would be recognized the contour or remnants of several larger vessels, completely obliterated, in a partly necrotic, extremely cellular tissue, smaller infiltrations extending farther along widened vessels with infiltrated walls. The infiltration consisted of rather large mononuclear cells, lymphocytes, plasma cells, eosinophile cells, but, as a rule, without any polymorphonuclear leukocytes, but in places would be cells with fat granules. Extending into the brain substance the infiltration was principally perivascular, consisting mostly of mononuclear cells. The most striking feature was the involvement of the medium-sized arteries in the sulci and of their branches into the brain, the veins being also involved but in less degree. All the coats of the arteries were thickened and infiltrated, the intima being so greatly thickened that the lumen might be completely obliterated. This change of the intima seemed to be due largely to proliferation of its connective-tissue elements, less to invasion of other cells. In some of the smaller arteries were found multinuclear giant cells, rather small, the nuclei partly central, partly peripheral, partly diffusely scattered over the cells, which occurred especially in the peripheral layer, and intima. In the most severe lesions the different layers of the arteries would disappear, leaving only the outlines of the vessels in the midst of necrotic granular material, but nowhere was there any arrangement like that in tuberculosis or definitely caseous foci. The involvement of the veins was less marked; in places the veins were thrombotic, and in larger necrotic areas they were also necrotic. Surrounding larger masses the small vessels of the brain were greatly dilated and congested.

It would seem that the brain lesion described in this case was the cause of the mental disturbances during life. It is more reasonable to take this view than to believe that in addition to the changes described there had existed also a separate and distinct mental disease. But the principal question is, What kind of arterial disease was this? The first thought was that it concerned a tuberculous process. The vascular infiltrations with the numerous giant and epithelioid cells might point in this direction, but real tubercles or typical tuberculous granulation tissue was not found and tubercle bacilli could not be demonstrated, nor was there any tuberculosis in other organs, hence we are forced to conclude that it does not concern a tuberculous process. Lethargic encephalitis is contraindicated both by the clinical symptoms, the localization and character of the lesions. In fact, no changes such as have been observed in the various stages of this disease were found in the case under consideration. The possibility of syphilis must be considered. There is the history of chancre in 1905; it is true that the Wassermann reaction of the blood serum was negative, but this of itself hardly excludes syphilis. The absence of syphilitic changes elsewhere do not necessarily exclude syphilis. The vascular process resembles in many ways syphilitic arteritis; proliferation of the intima resembles the syphilitic, although perhaps not so marked; the cellular infiltration in the other vascular coats also remind one of syphilis; and numerous giant cells do not exclude syphilis, although they are more numerous here than usual. Against syphilis we have the marked thickening and necrosis of the media and adventitia and the disintegration and destruction in places of the elastic tissue in the media. Spirochetes were not found, but it is difficult indeed to demonstrate spirochetes even in definitely syphilitic vascular processes. The localization is not inconsistent with syphilis, but a widespread process is rare in syphilis, and it would be expected that gummatous and caseous foci would be found in case it concerned syphilis, but they were absent. We see that while important arguments may be brought forward against syphilis it cannot be definitely excluded.

The localization speaks against periarteritis nodosa. With the exception of the cases described by Chvostek and Weikselbaum and Müller,[10] practically all descriptions of this disease state that the arteries of the brain are not involved. However, the lesion itself corresponds quite closely to that of periarteritis nodosa, the extensive necrosis of the media with separation and destruction of the elastic tissue being marked, but in many cases the necrosis was even more extensive than usual in periarteritis nodosa, and giant cells, which were very numerous in this case, are found only rarely in this disease, the principal changes of which, after a duration of say nine months, appear to be proliferation of the intima and media with necrosis and degeneration, with but little cellular infiltration in the vessel wall and no giant-cell formation (Gruber,[11] Abramof[12] and v. Haun;[13] see also my Case VII). In no case was there such marked necrosis as in my Case IV, and no giant cells, but numerous epithelioid cells. It remains to point out that there were no definite or typical aneurysms in this case. Taking everything into consideration it must be acknowledged that in many ways this case reminds one of periarteritis nodosa, but even more of syphilitic endarteritis.

For purpose of comparison I shall now describe a case of cerebral syphilis with vascular changes of a peculiar character and of special interest in this connection.

CASE V.—A man, aged forty-two years. According to Dr. S. Höyer he began to have pain in the left ear in July, 1900, and in September a discharge appeared from the ear and a little later the left side of the face appeared to be paralyzed. In October there was found suppurative otitis with a large defect in the membrane, but no tenderness over the mastoid. The forehead could be wrinkled but the left eye closed only slowly and incompletely. By the latter part of December the discharge ceased and the general condition was good. In February, 1901, he suffered some kind of injury, and headache and dizziness came on, together with increased discharge again from the ear. A few days later he became paralyzed suddenly in the right arm and the right lower extremity. In the latter part of March the paralysis was improved, but he seemed dull and complained of pain in the left side of the back of the head and also of dizziness. No choked disk. Radial artery thick and second aortic sound accentuated; pulse 80, unequal; left side of face smooth, no function of facial nerve; left lower extremity immovable; lively reflexes; both arms moved freely, grip strong, tremor; headache, frontal and occipital, drowsiness; external otitis (left). March 29 a complete mastoid resection was made, but nothing abnormal was found, and punctures of the brain did not reveal any pus. The patient became comatose after the operation and died two days later.

[11] Centralbl. f. Herz. u. Gesfasskrankh., 1917, No. 9.

[12] Ziegler's Beiträge, 1899, No. 26.

[13] Virchow's Arch., 1920, No. 227.

At the postmortem the tibiæ were found to be thick, the skin scarred and brown; on the inner side grayish-red subperiosteal granulation tissue in small holes in the surface of the bones, otherwise is hard and thick. Skin and skeleton elsewhere showed nothing abnormal.

The heart was a little large, the aorta presented a few small yellowish spots and a small scar between the arch and the descending part.

There was bronchitis and double bronchopneumonia; bronchial nodes calcareous.

Spleen weighed 220 grams; adherent to diaphragm. Liver normal. Kidneys showed some fatty change; bladder normal; right testicle fibrous throughout and contains a yellowish-white, putty-like mass as large as a pea; in the left testicle is a yellowish-white cord containing a small concretion. Digestive tract normal.

In the left temporal fossa was a reddish-brown exudate, and under the left side of the cerebellum. The right hemisphere seemed strongly injected, reddish, while the left was pale. Along the left Sylvian artery and its branches were small yellowish-white or yellowish-gray fibrous lines and spots. Pia arachnoid adherent over the right frontal lobe; the cortex here was peculiarly reddish violet in color. Pia arachnoid over the left hemisphere was free.

The vessels at the base were greatly changed, irregularly thickened and dilated and grayish-red in color. These changes were most marked in basilar artery and in Sylvian arteries, which on the left side showed thrombosis.

Nothing abnormal was found in the left hemisphere except a small softened focus between the caudate nucleus and the internal capsule and a little larger focus in the outer part of the lenticular nucleus. The right hemisphere was hyperemic and free from other changes. There was a thin veil-like layer around the medulla and the upper part of the cord; cord and its membranes otherwise normal.

Diagnosis. Syphilitic endarteritis of the cerebral arteries with thrombosis in the left Sylvian artery, softening in the left caudate and lenticular nucleus; meningo-encephalitis over the right frontal lobe, the left temporal lobe and medulla; hyperemia of the right hemisphere; fibrous and caseous syphilitic orchitis; syphilitic periostitis of the tibiæ; bronchitis and bronchopneumonia.

Microscopically the walls of the arteries showed an extensive, somewhat irregularly distributed, even purely nodular inflammatory process, often with great narrowing of the lumen. The adventitia was always the seat of an extensive round-cell infiltration and also in places of connective-tissue proliferation. The media showed degeneration of the muscle cells and of the elastic layer which were broken especially the internal elastic layer underneath foci in the intima containing giant cells. The intima showed the greatest changes: the cells had proliferated and formed compact masses which narrowed the lumen and even formed swellings of the wall. In places there was complete obliteration, oftenest due to secondary thrombosis. Giant cells, single and in masses, with peripheral nuclei and a degenerated center were found oftenest in the peripheral part of the intimal proliferation, sometimes forming tubercle-like nodules or miliary gummas. There were also foci with a great number of giant cells. A characteristic feature was numerous newly formed small vessels in the intima, and it seemed as if certain necrotic foci were due to obliteration of small vessels.

Besides the lesion in the arteries there were inflammatory changes in the membranes, more over the right hemisphere than the left, and most at the base, but present also over the medulla and the dorsal cord. The infiltration consisted of lymphocytes and some plasma cells and was directly connected with the changes in the adventitia, being often very marked around small arteries as well as veins, along which the infiltration extended into the cortex, which especially on the right side was quite hyperemic. On the inner side of the dura over the left temporal lobe was round-cell infiltration as well as fresh exudate.

This case is interesting first because of the difficulty in the way of a clinical diagnosis and next because of the extent and nature of the lesions in the brain, leptomeningeal and vascular. The endarteritis is remarkable because it occurred in the form of nodules in the region of the left Sylvian artery that often looked like aneurysms and reminded one of periarteritis nodosa. However, taking the case as a whole, there can be no question that it concerned syphilis.

The numerous giant cells in the granulation tissue in the walls of the smaller arteries with degeneration in the intima and media place this case in a rather unusual group of gummous endarteritis. Otherwise the endarteritis seems to correspond to the usual descriptions. It is true that formerly it was denied that giant cells occur in syphilitic vasculitis, but Wright[14] and others have shown conclusively that they may occur.

The microscopic picture excludes periarteritis nodosa, but it brings to mind the tuberculous form of vascular lesion, but no tubercle bacilli were found—there were no tuberculous processes anywhere else—and inoculations of guinea-pigs with the caseous material from the testicle had no effect.

CASE VI.—*Syphilitic Coronary Arteritis Resembling Tuberculosis* (Figs. 7 to 9). Some years ago I[15] studied a case of gummous syphilis of the heart in a man, aged thirty-eight years, who had had syphilis fourteen years before and who was sick for four days

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[14] Report of the Massachusetts General Hospital, 1909.

[15] Norsk. magasin f. Lägevidenskabeen, 1914.

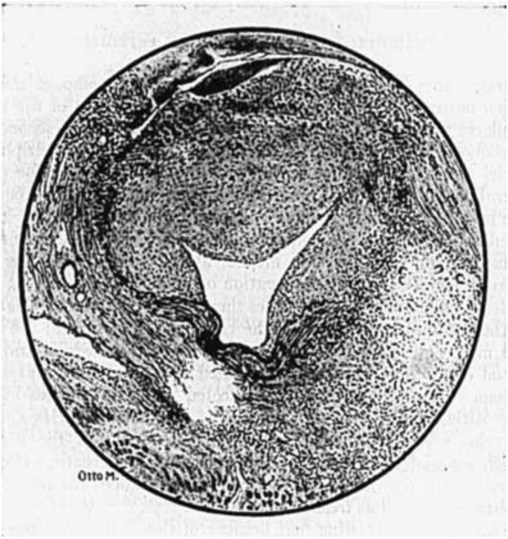

Fig. 7.—Extensive syphilitic endarteritis with necrosis and giant cells.

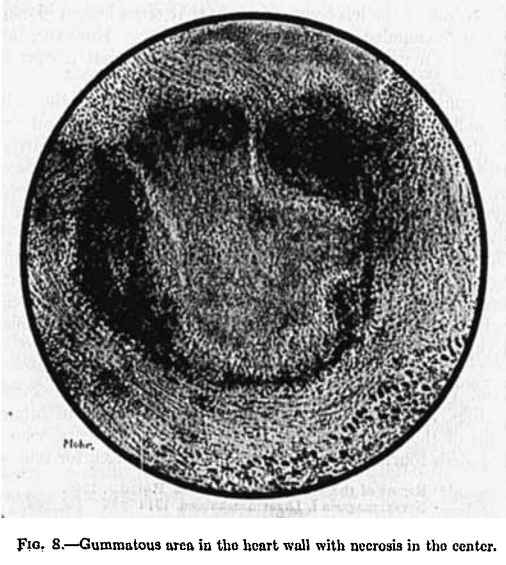

Fig. 8.—Gummatous area in the heart wall with necrosis in the center.

with dyspnea, cyanosis and rapid pulse. Gummas were found, particularly in the walls of the right ventricle and the atrium, which had resulted in obliteration of the superior vena cava. There was also a syphilitic endarteritis in the coronaries and a circumscribed gummous process in the pleura and adjacent parts of the left lung. In this case the changes in the walls of the vessels—necrosis, giant cells, etc.—resembled greatly tuberculosis, as shown by Figs. 7 to 9.[16]

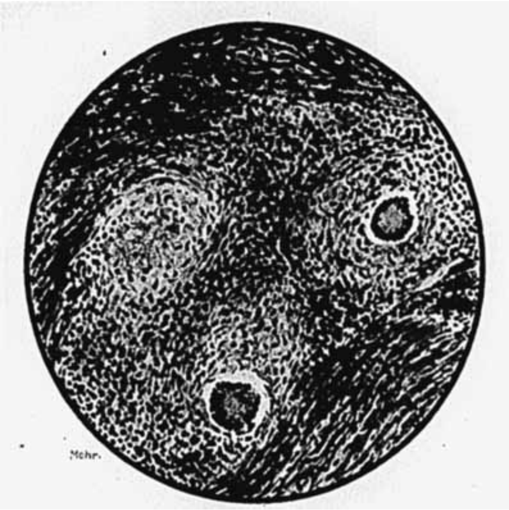

Fig. 9.—Inflammatory gummatous process in the heart wall; epithelioid cells and giant cells. The appearance in Figs. 7, 8, and 9 resemble tuberculosis.

Finally, I wish to give a brief summary of a typical case of periarteritis nodosa.[17]

[16] In Norway it was early pointed out that syphilis may be the cause of important diseases in the internal organs, particularly the brain, the heart and the kidneys. Thus Gjör in 1857 described thirty cases of cerebral syphilis early in life, including cases of apoplexy, hemoplegia and general paralysis. Furthermore, E. Winge in 1863 described changes in the internal organs in syphilitics, including a case of gummous aortitis and gummas in the heart with aortic aneurysm and a case of gummous spinal pachymeningitis. But it was particularly G. Heiberg who insisted on the importance of syphilis in vascular diseases; he demonstrated in the medical society in Christiania (see Transactions, 1876, 1877, 1886) numerous specimens of syphilitic arteritis and aneurysms.

[17] Ibid., 1917.

CASE VII.—*Periarteritis Nodosa* (Figs. 10 to 14). The patient was a man, aged twenty-two years, who began to suffer with stabbing pains in the left foot in April, 1916. Later he noticed that the left foot, which was tender, felt dead. It was easy for him to stumble. In the middle of May similar pains appeared in the right foot and leg and the condition in both feet became worse, at the same time as he had some fever. There was nothing of interest in the family history and the patient himself had never been seriously ill; no syphilitic infection. The cerebral nerves and the upper extremities were not involved. The lower extremities were normal in position, rather thin, no tremor, no rigidity, rather weak. The muscles in the legs and thigh were tender. The walk was somewhat paretic and he could not walk on his toes or sit on his feet. Wassermann reaction negative. In November palpitation of the heart appeared; the pulse was frequent with a pressure of 160; later dyspnea appeared also systolic murmur. The urine contained albumin and casts. There was also tenderness in the epigastrium with vomiting and a slight icterus. In December edema appeared about the ankles and scrotum, also diarrhea, and the dyspnea increased, there being also

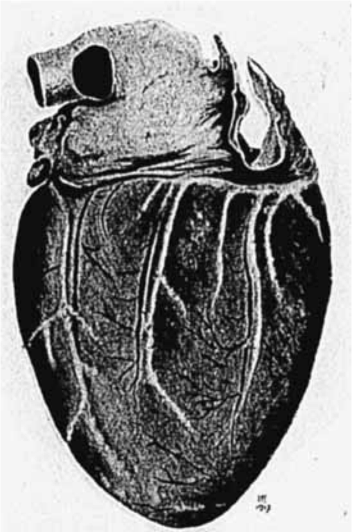

FIG. 10.—Heart from the left side with numerous nodules on the small arteries.

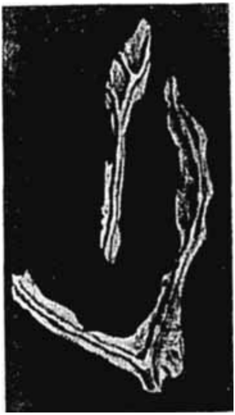

FIG. 11.—Arteries from the omentum with numerous small nodules.

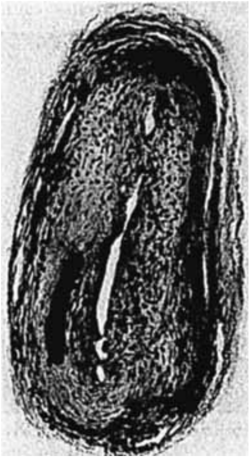

FIG. 12.—Cross-section of the coronary branch, showing infiltration of the intima with calcareous deposit, partial destruction of the media and infiltration of adventitia.

cough with bloody expectoration. The edema increased, ascites developed, the vomiting became worse and the vomited matter contained blood. Death occurred in January, 1917.

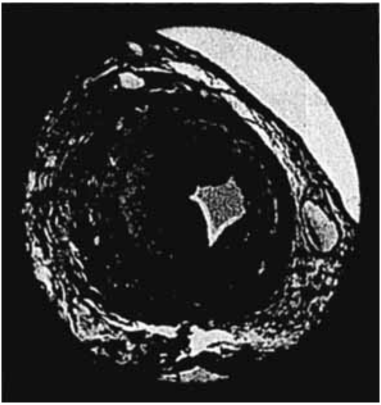

FIG. 13.—Photomicrograph of the artery in the omentum. × 150.

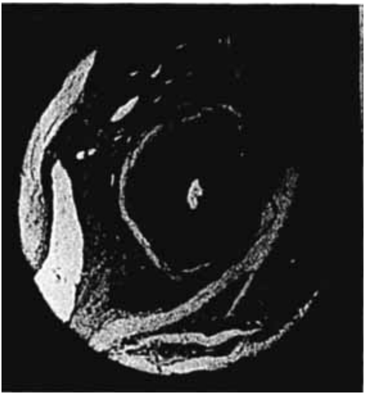

FIG. 14.—Photomicrograph of the arterial branch in the muscle of the leg. × 70.

The postmortem examination showed altogether normal conditions in the brain and its membranes; the arteries at the base showed no changes.

The heart with 570 grams, and as shown in Fig. 10, the coronaries, which were very prominent, showed here and there oval grayish-white nodules up to the size of a hempseed, most numerous along the left side and posteriorly. In the muscles were peculiar yellowish-white spots and stripes as well as hemorrhagic infiltration, and also fibrous streaks. The heart valves were normal.

Lungs and pulmonary arteries were normal; hydrothorax. The organs of the neck and mouth were normal.

There was some ascites. The spleen contained an anemic infarct and three or four small yellowish-white spots. The liver weighed 1040 grams, fatty and passively congested. The vessels on the surface appear as white, thick strings, especially marked on the anterior surface of the right lobe.

The kidneys weighed 310 grams; on the surface numerous irregularly distributed bluish-red, slightly depressed infarct-like areas; on the surface the vessels were more prominent than usual and thicker; on the border between the cortex and the medulla are some grayish-white nodular prominences.

Adrenals and pancreas normal.

On the surface of the stomach the arteries are distinct and here and there are pinhead-sized nodules on the smaller branches. In the mucosa of the smaller curvature near the pylorus is a small ulcer and near it is a small nodule on a little arterial branch. There is a hemorrhagic erosion in the fundus. In the ileum the membrane is hemorrhagic, the follicles prominent; the large intestine showed a swollen lining with hemorrhagic infiltration and necrosis here and there.

The arteries in the omentum are nodular throughout.

The aorta is a little uneven and thick along the posterior wall; this is also true of the iliacs, but the large abdominal arteries are normal.

The spinal cord seems normal, also the vagus, phrenic, brachial, ulnar and sciatic nerves, but there was some edema and small hemorrhages in the connective tissue and muscles about the latter three nerves.

The microscopic examination of the smaller arteries in the omentum, the stomach, the heart, the kidneys and the spleen showed marked swelling of the media and intima in places, due principally to a hyaline degeneration of the connective tissue, in which were only few fibroblasts and here and there some calcareous infiltration. The change was most marked in the intima. In the media the muscle fibers and elastic layers were degenerated but not necessarily all around the lumen. The thickening led to narrowing and even complete obliteration of the lumen and also irregular nodules, so-called aneurysms, which were solid. These changes were most marked in the arteries of the heart and kidneys. In the adventitia there were only slight cellular infiltrations. Similar changes were found in the small branches of the popliteal, in the branches of the brachial plexus and in the median and sciatic nerves with surrounding muscle tissue, and in this case there was also considerable infiltration in the connective tissue around the arteries and in the nerves, mostly of lymphocytes.

In the heart muscles was infiltration of the lymphocytes, increased in fibrous tissue, and degeneration with fragmentation of the muscle fibers with calcareous dust in places. In the kidneys degenerative and inflammatory changes as well as infarcts were present in addition to the arterial changes.

In the other organs there were no special microscopic changes other than those indicated by the naked-eye examination.

Summarizing the changes it may be noted that the smaller arteries in the heart, the kidneys, the stomach, the intestine, the omentum and in certain muscles and nerves were the seat of a productive inflammatory process, particularly in the media and intima; in the arteries in the muscles and nerves there was also an exudative process, particularly in the adventitia.

Periarteritis nodosa presents different clinical forms according as different organs are involved. It begins usually acutely in previously well persons and attacks mostly young and strong men between twenty and forty years of age, the first symptoms as in this case being indefinite pains in the muscles of the trunk as well as of the extremities and in the joints with fever; there may be edema and changes in the skin with disturbances in sensibility, paresis, exhaustion and weakness, the pulse often being very frequent and irregular. Death usually occurs after some weeks up to six or eight months after increasing weakness, dyspnea, renal and digestive disturbances as well as cardiac troubles. Death has been observed as early as ten days from the beginning and as late as after one or two years. The most common form is the muscle-nerve type, to which the case I have described belongs. In other cases the principal symptom may be renal or gastro-intestinal or cardiac; other organs, *e. g.*, the skin, may be especially involved, but as before stated the brain is rarely affected. The disease appears to be curable —four such cases have been described—and it may well be that more instances of recovery of mild or abortive forms will be recognized as the disease becomes better known. Most of the cases—between sixty and seventy in all—have been described in German literature and only remarkably few in the English and American.

The primary anatomical changes are in the small and medium-sized arteries in the heart, kidneys, digestive tract, etc., where they give rise to the very characteristic oval grayish-white, usually firm aneurysm-like nodules in linear arrangement on the small arteries. The lumen is usually narrowed, but true aneurysms have been observed. The large arteries, as a rule, are not involved. The narrowing and thrombosis of the arteries form the basis for the other anatomical changes as well as the clinical phenomena; necroses and degenerations, hemorrhages and hemorrhagic infiltrations.

Microscopically the inflammatory changes in the arteries appear to be infectious. There is no agreement as to whether the changes develop first in the media or adventitia, although many believe that the media is first involved, fibrinous exudation and edema with degenerative and necrotic changes in the muscles fibers and elastic tissue, together with round-cell infiltration preceding the formation of connective tissue. By some the primary lesion is believed to be a periarteritis which extends into the media and involves the intima in proliferation. When the changes are acute true aneurysms may form.

Such in brief appears this disease of the arteries. It has many things in common with the so-called syphilitic endarteritis, but there are also differences. As emphasized by Guldner,[18] the presence of spirochete and a positive Wassermann reaction would establish the diagnosis of syphilis, which frequently involves the cerebral arteries while periarteritis nodosa rarely occurs in the brain; in syphilis the intima is first involved, whereas in periarteritis the media or the adventitia; in syphilis gummatous changes and giant-cell formation occur while the latter is rare in periarteritis nodosa; and finally in the syphilitic endarteritis the elastic membranes are split apart and new elastic tissue may be formed, but there is no such fracturing of the elastic layers as in periarteritis. Spiro[19] points out that the infiltration in periarteritis regularly contains polymorphonuclear leukocytes, which are absent in syphilitic endarteritis, and the vessel wall is attacked only partially in periarteritis, whereas in syphilis the process usually extends all around the vessel. It should be noted that in syphilitic endarteritis the intima may be attacked secondarily; that the giant-cell formation is rather unusual in both these forms of arterial disease, although more so in periarteritis nodosa; and the necrosis in periarteritis nodosa may be quite considerable even though large gummatous areas do not form. There seems to be no absolutely distinctive difference between these two diseases of the arteries, and consequently it is not difficult to understand that they may be confused. Versé[20] has advanced the opinion that they may be identical because they have so many things in common; thus aneurysms may develop in syphilitic endarteritis and necroses are present in both processes as well as giant-cell formation, and he suggests that periarteritis nodosa may be a special form of arterial syphilis affecting small and medium-sized arteries—but if only a few of the cases of periarteritis nodosa has syphilitic infection been definitely demonstrated and, as a rule there is no history of syphilis and the Wassermann reaction is negative. While it is true that the lesions in these two diseases in many respects corresponds in particular details, the complete anatomical picture seems to me to indicate that it concerns two distinct diseases.

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[18] Virchow's Arch., 1915, No. 219.

[19] Ibid., 1920, No. 227.

[20] Ziegler's Beiträge, 1907, No. 40.

Certain writers hold that periarteritis nodosa is not a distinct entity because no specific virus as yet has been demonstrated and because various pyogenic microbes have been found in some of the cases (staphylococci, streptococci;) it is also pointed out that preceding infections such as angina, typhoid fever, diphtheria and acute rheumatism may be regarded as predisposing, in some cases at least, to the development of periarteritis nodosa. It is true, as discussed in the earlier part of this article, that arteritis occurs in different forms of infection, but at present the view that the disease is not specific may be regarded as merely hypothetical. To me it seems most reasonable to regard periarteritis nodosa as a definite disease due to a distinct virus. While the clinical picture may be variable, depending on the distribution of the lesions of the arteries, the lesion itself is quite characteristic enough to warrant the belief that it concerns a specific disease. Recently, v. Haun[21] by inoculation of the blood of a patient with periarteritis nodosa claims to have produced the disease in guinea-pigs, but of course further investigation is required.

In conclusion I would point out that forms of true arteritis occur more frequently than usually believed, that they apparently differ etiologically, and that they present anatomical appearances that make the differential diagnosis difficult, this being particularly true of the vascular changes in syphilis, periarteritis nodosa and also in tuberculosis.

POSTOPERATIVE DIETOTHERAPY.[1]

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and

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Prior to the middle of the nineteenth century the dietetic treatment of most acute disease conditions consisted in virtual starvation. Graves, in 1848, first insisted that a fever patient should be fed. Today clinicians are in accord regarding the dietary treatment of febrile conditions. The high calorie treatment of typhoid fever is a

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[21] Virchow's Arch., 1920, No. 227.

[1] Read before the Richmond Academy of Medicine, Richmond, Va., February 22, 1921.